Journal of Forensic and Legal Medicine 16 (2009) 253–260

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/jflm

Original Communication

Pathoanatomy of the lower cervical spine facet joints in motor vehicle crash fatalities

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ARTICLE INFO

Article history:
Received 30 January 2008
Received in revised form 2 September 2008
Accepted 9 December 2008
Available online 23 January 2009

Keywords:
Autopsy
Microscopy
Facet joint
Road traffic crash
Post-mortem
Cervical spine

SUMMARY

Non-lethal injuries to the cervical spine facet joints have previously been described in decedents from motor vehicle crashes and in clinical settings these joints have been identified as potential culprits in chronic neck pain syndromes. The aim of this study was to conduct a detailed examination of the lower cervical spine facet joints in a forensic cohort of motor vehicle crash victims and controls using comparable data from medicolegal autopsy, stereomicroscopy and histological evaluations. Injuries to the cervical spine facet joints were common in the trauma cases and included facet fractures, haemarthrosis, and disruption and bleeding in the synovial folds. The injuries could not be reliably verified on stereomicroscopic evaluation, and routine autopsy procedures did not reveal any of the injuries to the facet joints. Despite the presence of these pathoanatomical lesions in road traffic crash fatalities their prevalence and potential clinical implications in survivors from motor vehicle crashes is unknown.

1. Introduction

Road traffic crashes are responsible for the death of more than 40,000 people in Europe annually, and in addition to the fatalities there is an estimated 3.5 million casualties per year in Europe with an estimated cost of 250 billion Euro.[1,2] Non-fatal injuries to the cervical spine facet joints have been related to the clinical picture of chronic neck pain following road traffic crashes which is responsible for substantial morbidity.[3–6] However, the aetiology of chronic neck pain with regard to which somatic structures may be injured, damaged or diseased remains unclear.

Previous post-mortem studies have examined road traffic crash victims for the presence of cervical spine injuries that are not apparent on a range of diagnostic imaging modalities but revealed with cryomicroscopy and/or histological examination. The injuries detected include injuries to the facet joint capsules, synovial folds and articular surfaces.[7–12] Only few studies, however, have evaluated homogenous study groups with regard to detailed evaluation of the mechanism of trauma, cause of death and microscopical findings and compared these to findings in control group subjects, and the prevalence of discrete facet joint injuries following fatal road traffic crashes is unknown.

Biomechanical studies of cadavers in which the cervical spine has been exposed to relatively low acceleration forces (3.3–4.5*g*) followed by cryomicroscopical examination have reported similar pathological findings to those identified in the road traffic crash victims, indicating that injury thresholds for spinal injury can be superseded in a range of crash conditions.[13]

In clinical settings diagnostic imaging studies of survivors from road traffic crashes of a wide range of severities are often negative despite subjective complaints from the patients. However, not withstanding advancements in the field of diagnostic imaging procedures, including improved resolution of computed tomography and magnetic resonance imaging, discrete injuries may remain undetected on these investigations.[14] Hence, if real injuries are not always apparent with imaging in crash injury survivors this raises the possibility that such injuries will go unrecognized and untreated, and that the patients may be considered malingerers or frauds.

For the purpose of examining the prevalence of discrete injuries in the human cervical spine facet joints following fatal road traffic crashes a forensic cohort can be examined which is very likely to

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doi:10.1016/j.jflm.2008.12.014

Table 1
Detailed description of the autopsy findings.

Subject	Age	Gender	Circumstances	Primary cause of death	Skull fracture	Cervical spine vertebral fracture	Thoracic spine, lumbar spine and/or pelvic fractures	Extremity fractures	Intracranialorgan injuries	Thoracic organ injuries	Abdominal organ injuries
13	34	M	Driver, RTC	Bleeding, multiple traumatic injuries	No	No	Pelvis	Humerus, femur, tibia, fibula	Yes	Yes	Yes
14	20	M	Driver, RTC	Multiple traumatic injuries	No	C1, C2	Th3, Th4	No	Yes	Yes	No
17	45	F	Driver, RTC	Haemorrhagic mediastinum/rupture vena cava	No	No	No	Tibia, pedis	No	Yes	Yes
19	35	M	Driver, RTC	Subarachnoid haemorrhage/cerebral contusion	No	No	No	Ulna, radius, femur, tibia, fibula	Yes	No	Yes
20	29	M	Driver, RTC	Multiple traumatic injuries	Yes	No	Pelvis	Femur	No	Yes	Yes
21	31	F	Driver, RTC	Cerebral contusion/ medulla oblongata lesions	Yes	C0–1,C2–3	No	Tibia, fibula	Yes	Yes	Yes
22	20	M	Driver, RTC	Pulmonary contusion and laceration	No	No	Th9	No	No	Yes	Yes
24	22	F	Driver, RTC	Multiple traumatic injuries	Yes	No	No	Ulna, femur and tibia	Yes	Yes	Yes
25	36	M	Driver, RTC	Multiple traumatic injuries	No	C1, C6-7	No	Femur	Yes	Yes	Yes
26	21	M	Passenger, RTC	Multiple traumatic injuries	Yes	No	Pelvis	Humerus, femur	Yes	Yes	Yes
28	38	M	Driver, RTC	Bleeding and multiple traumatic injuries	Yes	No	No	Femur, calcaneus	Yes	Yes	Yes
34	47	M	Driver, RTC	Bleeding and aortic lacerations	No	No	Th11, Th12	No	No	Yes	No
35	33	M	Driver, RTC	Bleeding, haemothorax, rupture thoracic aorta	No	No	No	Tibia, pedis	No	Yes	Yes
36	29	M	Driver, RTC	Multiple traumatic injuries	Yes	No	Th5–6	Humerus, ulna, tibia, pedis	Yes	Yes	Yes
39	41	M	Driver, RTC	Haemothorax, aortic rupture	No	No	Pelvis	Tibia	Yes	Yes	Yes
42	29	M	Driver, RTC	Bleeding, injuries to the heart and aorta	No	No	No	Femur, tibia, fibula	No	Yes	Yes
46	37	M	Driver, RTC	Multiple traumatic injuries	Yes	C1–2	Pelvis	No	Yes	Yes	Yes
47	23	F	Driver, RTC	Bleeding, multiple traumatic injuries	No	No	No	Humerus	No	Yes	Yes
53	31	M	Driver, RTC	Multiple traumatic injuries	Yes	No	Th12, pelvis	No	No	Yes	Yes
19 cases					8	4	10	14	11	18	17
12	41	M	Vital activity	Myocardial infarct	No	No	No	No	No	No	No
15	32	M	Leisure activity	Unknown	No	No	No	No	No	No	No
16	39	F	Vital activity	Myocardial infarct	No	No	No	No	No	No	No
18	37	M	Vital activity	Epileptic seizure/ subarachnoid inflammation	No	No	No	No	No	No	No
23	37	M	Other activity	Cardiac disease	No	No	No	No	No	No	No
29	34	F	Vital activity	Unknown, intoxication (alcohol)	No	No	No	No	No	No	No
30	43	F	Surgery	Cardiac dysfunction secondary to surgery	No	No	No	No	No	No	No
31	31	M	Vital activity	Dehydration, peritonitis, paralytic small bowel	No	No	No	No	No	No	No

have suffered such injuries and where the injuries themselves have a remote relation to the cause of death.

The aim of this study was to examine the lower cervical spine facet joints in a population of people killed in a passenger car crash compared with an age-matched control group for the presence of cervical spine facet joint injuries using comparable data from medicolegal autopsy, stereomicroscopy and histological evaluations using the histological findings as gold standard.

2. Methods

2.1. Materials

Forty-two subjects were examined at medicolegal autopsy within 1–5 days (median 3 days) after death and included in this study. During autopsy the lower four cervical vertebral segments (C4–C7) including paravertebral muscles were removed *en bloc*, from twelve females (median age 40 years, range 22–49) and 30

Table 2
Detailed description of the toxicological findings.

Subject	Age	Gender	Blood alcohol concentration (‰)	Drugs/medicine in blood/urine
13	34	M	0	Ketamine[c]
14	20	M	0.88	No
17	45	F	0	Not tested
19	35	M	1.89	Not tested
20	29	M	0.10	Not tested
21	31	F	0	No
22	20	M	0	Valproate
24	22	F	0.00	No
25	36	M	0.90	Metoprolol
26	21	M	2.08	No
28	38	M	0	Not tested
34	47	M	0	Citalopram
35	33	M	0	No
36	29	M	0	Not tested
39	41	M	(2.40)[a]	Not tested
42	29	M	0	Not tested
46	37	M	0	Not tested
47	23	F	0	Not tested
53	31	M	0.22	Not tested
19 Cases			5 (BAC > 0.5)	4
12	41	M	0.78	Not tested
15	32	M	0	No
16	39	F	0	Not tested
18	37	M	0	No
23	37	M	0	Not tested
29	34	F	1.80	Cannabis[c]
30	43	F	0	Clomipramine & paracetamol[b]
31	31	M	0	Not tested
33	35	M	0	No
37	49	F	Not tested	Not tested
38	49	M	0	Not tested
40	27	F	0	Cocaine[c]
43	33	M	0	Not tested
44	40	M	0.28	Not tested
45	35	M	0	No
48	46	F	1.82	No
49	41	F	0	No
50	41	F	0	Not tested
51	23	M	0	Not tested
52	48	M	0	Not tested
54	22	M	0	No
21 controls			3 (BAC > 0.5)	3

BAC: blood alcohol concentration.
Gender: M (male), F (female).
[a] Conc. in eyefluid (vitrous humor).
[b] Conc. of intoxication (overdose).
[c] Narcotics/drugs.

33	35	M	Vital activity	Arteriosclerotic cardiac disease	No	No	No	No	No	No	No	No	No
37	49	F	Vital activity	Myocardial infarct	No	No	No	No	No	No	No	No	No
38	49	M	Vital activity	Arteriosclerotic cardiac disease	No	No	No	No	No	No	No	No	No
40	27	F	Unknown	Cardiac disease, diabetes mellitus, intoxication	No	No	No	No	No	No	No	No	No
43	33	M	Other activity	Bleeding due to cutting injury	No	No	No	No	No	No	No	No	No
44	40	M	Other activity	Cardiac disease	No	No	No	No	No	No	No	No	No
45	35	M	Unknown	Diabetic coma	No	No	No	No	No	No	No	No	No
48	46	F	Hypothermia	Hypothermia and intoxication (alcohol)	No	No	No	No	No	No	No	No	No
49	41	F	Vital activity	Cardiac disease	No	No	No	No	No	No	No	No	No
50	41	F	Vital activity	Subarachnoid haemorrhage/cerebral aneurysm	No	No	No	No	No	No	No	No	No
51	23	M	Vital activity	Diabetic coma	No	No	No	No	No	No	No	No	No
52	48	M	Working	Arteriosclerotic cardiac disease	No	No	No	No	No	No	No	No	No
54	22	M	Vital activity	Aortic aneurysm rupture and cardiac disease	No	No	No	No	No	No	No	No	No
21 controls					0	0	0	0	0	0	0	0	0

Gender: M (male), F (female).
RTC: road traffic crash.
Vital activity: sleep, rest.

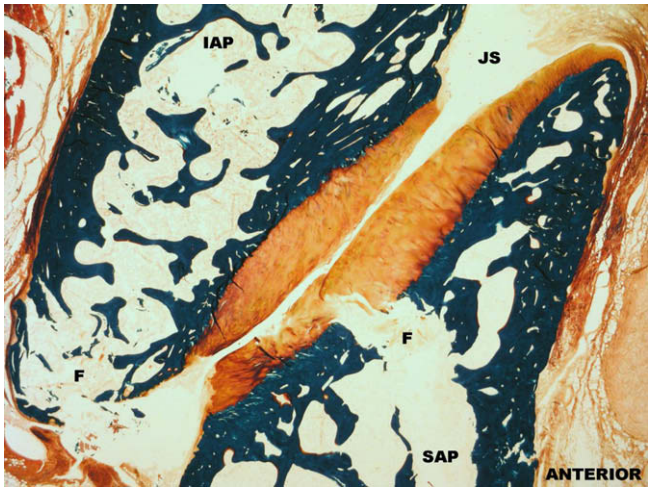

Fig. 1. Fractures of the opposing cervical spine facets in a motor vehicle crash fatality: overview of the joint, inferior articular process (IAP), superior articular process (SAP), joint space (JS) and facet fracture (F), original magnification ×1.25, Masson Goldner-Trichrome.

males (median age 34.5 years, range 20–49 years). Twenty subjects died in a passenger car crash (cases) and 22 subjects died due to non-traumatic causes (controls), and none of the decedents had died due to lower cervical spine injury. Subjects were excluded if there was evidence of previous history of drug and/or alcohol abuse, previous cervical spine injury, and extensive post mortem decomposition. After completed inclusion two subjects (one case and one control) were excluded from statistical analysis due to extensive decomposition causing generalized tissue damage. The study was approved by the local scientific ethics committee.

2.2. Methods

The specimens were fixed in 70% ethanol for a minimum of five weeks with hemisection in the median plane after two weeks, followed by one week in 96% ethanol under vacuum conditions fixation and a final week in 99% ethanol. Then each specimen was embedded in liquid methylmethacrylate (MMA) and dibutylphtalate (a softener) in a refrigerator for six weeks, after which percadox (a catalyst) was added to complete polymerisation and hardening into a plastic bloc. From a random central starting point, each plastic bloc was sawed by serial sectioning, into approximately seven 3-mm thick parasaggital slices using a precision guided bandsaw (Femi©, Bologna, Italy), which ensured exact thickness of the slices with a tissue loss of approximately 1 mm per slice. Each 3-mm thick slice was examined using an Olympus© stereomicroscope and scored with reference to damage of the facet cartilage and/or bone, bleeding in a joint, the presence of synovial folds and injury to the folds.

The 3-mm thick slices containing facet joints were re-embedded in MMA. Using a heavy-duty microtome (SM 2500, Leica©) two consecutive histological sections of 10 μm thickness were produced from each bloc, stained with Masson Goldner-Trichrome, and mounted un-deplastified on glass slides. All facet joints on histological sections were examined by light microscopy using a BX51, Olympus© microscope. An average of 45.8 unique observations per subject (2.9 observations per unique facet) were made including the scoring of predetermined variables; articular facet fractures, bleeding in the joint space, bleeding in the anterior and posterior synovial folds, and disruption (loss of normal structural integrity) of the anterior and posterior folds.

Fisher's exact test was used to examine the significance of association between categorical variables and Chi-squared test was used to examine the correlation of the number of disrupted folds per subject (case/control) versus exposure to trauma. Agreement between diagnostic methods was examined with kappa statistics. The significance level was $p < 0.05$. All statistical analyses were performed using Stata© 9 (StataCorp LP, College Station, USA).

3. Results

3.1. Autopsy findings

The medicolegal autopsy identified no injuries to the lower cervical spine facet joints (Table 1). In the trauma group seven unique

Table 3
Histopathological findings in the road traffic crash fatalities.

Subject #	Fracture of a facet	Blood in any joint space	Blood in # of ant. folds (0–8)	Blood in # of post. folds (0–8)	Anterior fold disrupted (0–8 joints)	Posterior fold disrupted (0–8 joints)
13	No	No	0	1	2	8
14	No	Yes	0	1	3	6
17	No	No	0	0	0	2
19	No	Yes	0	0	1	4
20	No	No	0	0	0	0
21	No	No	5	0	7	6
22	Yes	Yes	5	4	8	7
24	No	No	2	0	0	1
25	Yes	Yes	8	5	8	3
26	No	No	0	0	3	7
28	No	No	0	0	2	5
34	No	Yes	2	4	1	3
35	No	No	0	0	1	4
36	No	Yes	4	0	5	7
39	Yes	Yes	5	1	6	5
42	No	No	0	0	0	1
46	No	Yes	3	0	3	2
47	No	Yes	1	4	3	5
53	Yes	No	0	0	3	7
19 cases	4 ($p < 0.05$)	9 ($p < 0.01$)	35 (23.3%)[a] ($p < 0.001$)	20 (13.3%)[a] ($p < 0.001$)	56 (37.3%)[a] ($p < 0.01$)	83 (55.3%)[a] ($p < 0.01$)

Cases: 150 anterior and 150 posterior folds (19 subjects).
Controls: 168 anterior and 168 posterior folds (21 subjects).
[a] Percentage of all folds in the groups (two folds are non-existing due to a bloc vertebrae in one trauma case).

fractures in cervical spine vertebral bodies were identified in four trauma subjects (4/19 cases), with one classified as a disco-vertebral avulsion fracture at C6–C7, and the remaining fractures situated at or above C3. Skull fractures were identified in eight cases. There were significant injuries to cranial organs in 11 cases, thoracic organs in 18 cases, and abdominal organs in 17 cases. Injuries to the spleen and liver were affected in 15 cases and injuries to the lungs were affected in 15 cases. The primary cause of death in the trauma group was multiple injuries in 10 cases and bleeding in six cases (Table 1). In 18 of the cases the decedent was the driver of a passenger car and the remaining one case was a front seat passenger in a motor vehicle.

In the 21 control group subjects there were neither injuries to the musculoskeletal system nor traumatically induced organ injuries. The most common causes of death among the control group subjects was cardiovascular disease in 12 subjects.

Toxicological evaluation revealed a blood alcohol concentration (BAC) > 0.5 mg/ml in three drivers and one front seat passenger. In one case no blood was available for analysis, however the alcohol concentration in the vitreous humor was 2.4 mg/ml. Three control subjects had a blood alcohol concentration >0.5 mg/ml. All subjects except one control subject were tested for alcohol concentration. Medication was found in three cases (3/9 cases were tested) and one control (1/10 controls were tested) and narcotics were found in one case (1/9 cases were tested) and two controls (2/10 controls were tested) (Table 2).

3.2. Stereomicroscopy of the 3-mm thick slices

On the stereomicroscopy of the 3-mm thick slices damage to the facet cartilage and/or bone was present in 10 subjects (3/19 cases and 7/21 controls) and the presence of damage could not be evaluated in 29 subjects (15/19 cases and 14/21 controls). There was no significant correlation between exposure of trauma and the presence of damage to the facet cartilage and/or bone ($p = 0.28$). Bleeding in a joint was present in three subjects (3/19 cases), which did not correlate with exposure to trauma ($p = 0.10$), and the presence of bleeding in a joint could not be evaluated in 26 subjects (12/19 cases and 14/21 controls). Synovial folds were found in 22 subjects (22/40), they were absent in two subjects (2/40) are could not be evaluated in 16 subjects (8/19 cases and 8/21 controls) and no injuries to the folds were detected.

3.3. Light microscopy of the 10 μm thick sections

Light microscopy of the 10 μm sections identified facet fractures in four subjects (4/19 cases) (Fig. 1), and the presence of a facet fracture correlated with the exposure to trauma ($p < 0.05$) (Table 3). Bleeding in any joint space (haemarthrosis) was present in nine cases (9/19) and one control subject (1/21) (Fig. 2), and there was a significant correlation between bleeding in any joint space and the exposure to trauma ($p < 0.01$), between bleeding in any joint and the presence of a fracture on histological evaluation ($p < 0.05$).

Bleeding in an anterior fold was present in 35 of the folds from case subjects (35/150) and 10 from control subjects (10/168) with a risk ratio of 4 (95% confidence interval: 2–8, $p < 0.001$). Bleeding in a posterior fold was present in 20 of the folds from case subjects (20/150) and one from control subjects (1/168) with a risk ratio of 22 (95% confidence interval: 3–165, $p < 0.001$) (Fig. 3).

Disruption of an anterior fold was present in 56 of the folds from case subjects (56/150) and 23 from control subjects (23/168) with a risk ratio of 2.7 (95% confidence interval: 1.8–4.2, $p < 0.001$). Disruption of a posterior fold was present in 83 of the folds from case subjects (83/150) and 48 from control subjects (48/168) with a risk ratio of 1.9 (95% confidence interval: 1.5–2.6, $p < 0.001$) (Table 3). It is worth noticing that none of the injuries to the facet joints were observed during the autopsy.

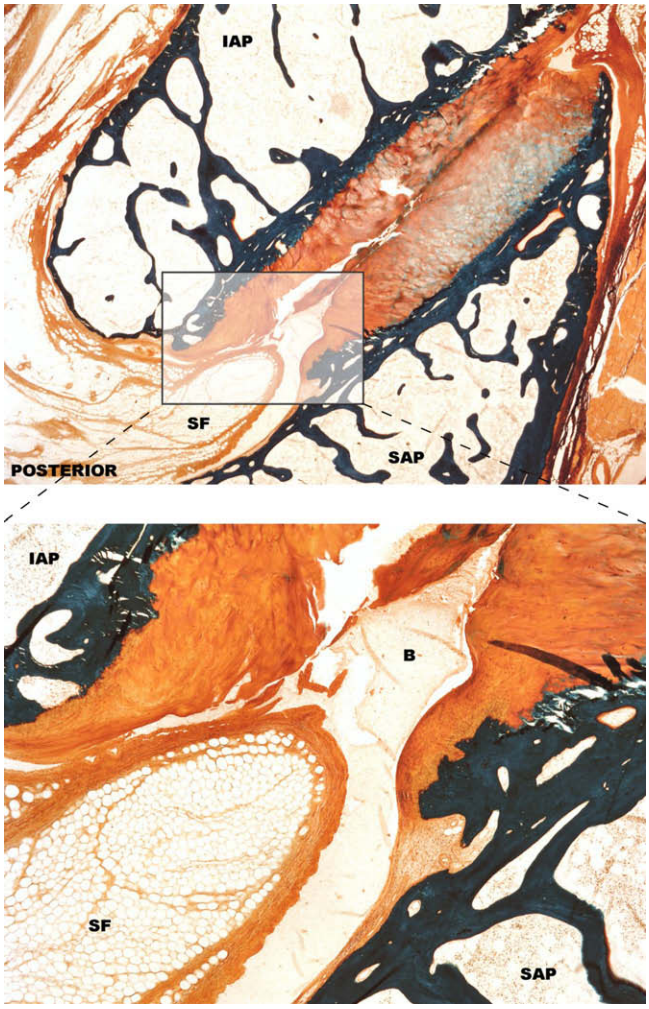

Fig. 2. Haemarthrosis in a cervical spine facet joint in a motor vehicle crash fatality: large overview of the joint, inferior articular process (IAP), superior articular process (SAP), synovial fold (SF), joint space (JS), original magnification ×1.25, Masson Goldner-Trichrome, and a close-up of shaded area illustrating bleeding in the joint space (B), original magnification ×4, Masson Goldner-Trichrome.

There was no correlation between the stereomicroscopy and histological findings concerning damages to the facet cartilage and/or bone or the synovial folds.

4. Discussion

The detection of non-fatal injuries to the cervical spine facet joints was not reported equally among the diagnostic methods used in this study. Conventional autopsy allowed detailed description of injuries, however no injuries to the cervical spine facet joints were reported. Results from the stereomicroscopic evaluation of the 3-mm slices did not correlate statistically with any of the parameters evaluated. In contrast, light microscopical evaluation of the 10 μm thick sections revealed statistically significant correlations between exposure to trauma, and the presence of non-fatal articular facet fractures, bleeding in the facet joints, bleeding in the folds, and disruption of the folds.

In this study the most common cause of death in the trauma group was multiple injuries, and the high incidence rates of skull fractures and organ injuries were all in concurrence with previously reported injuries in road traffic crash fatalities.[15] Forensic toxicology

revealed that four of the 18 drivers killed were under the influence of alcohol during the time of the crash which is similar to previously reported figures of 20–30% of decedents from fatal road traffic crashes having BAC values above 0.5 mg/ml.[16–18] Similarly, one trauma case tested positive for drugs which also supports previous reports of drugs in road traffic crash fatalities.[17] The medicolegal autopsy did not identify injuries in the cervical spine facet joints despite histological and neuroradiological evidence of injuries. However, this is likely due to the choice of autopsy technique utilized (i.e. anterior approach), which does not allow detailed description of the posterior elements of the cervical spine. Hence, the poor sensitivity towards identifying injuries in the facet joints is not surprising and concurs with other studies reaching the same conclusions regarding routine autopsies.[7,8,11,12,19]

Several post-mortem studies of trauma fatalities have reported subjects with fractures of the lower cervical spine facet joints similar to those identified in our study.[7–9,11,19–21] However, focusing on studies examining road traffic victims, the one in 22 and one in 15 traffic accident victims sustaining lower cervical spine facet joint fractures, examined in the studies by Jónsson et al.[9] and Taylor and Twomey,[7] respectively, is significantly lower than our findings of facet fractures in 4 of 19 trauma cases. The differences between the reported figures may be due to the primary focus on facet joint injuries in our study in contrast to the previous studies examining the whole cervical spine. Furthermore, our material exclusively involved subjects killed in motor vehicle crashes which indicate that all the decedents have been exposed to forces of significant magnitudes. The low number of subjects examined in all the studies as well as technical issues such as the histological methods utilized could influence the results.

Haemarthrosis of the cervical spine facet joints have been described in post-mortem studies of road traffic crash victims using histological and/or microtomal methods.[7–9,11,19,20] However, due to the nature of the different methods used in these studies the presence of erythrocytes in the joint cavities have not been clearly demonstrated in all studies, in particular not in studies utilising photographic documentation of the remaining tissue bloc after removal of micrometer thick sections. In the current study the erythrocytes were, after and in comparison with blood vessel content, identified as pale cell ghosts in a honeycomb pattern. By this definition, haemarthrosis was identified in almost half the trauma cases with statistical significance. Similarly, a recent study investigated 10 consecutive trauma fatalities, with no reference to the proportion of traffic crash victims in the material and the specific segments examined, and found haemarthrosis in the facet joints in 6 of the 10 cases based on microscopy of 3-mm thick slices and in some cases histopathologic examination.[19] In another post-mortem study haemarthrosis was present in six of 15 trauma cases, with only three affecting the lower cervical spine facet joints below C4.[7] However, the exact manner of identification of bleeding within the joints was not described in detail in any of these studies and combined with our finding that stereomicroscopic evaluation

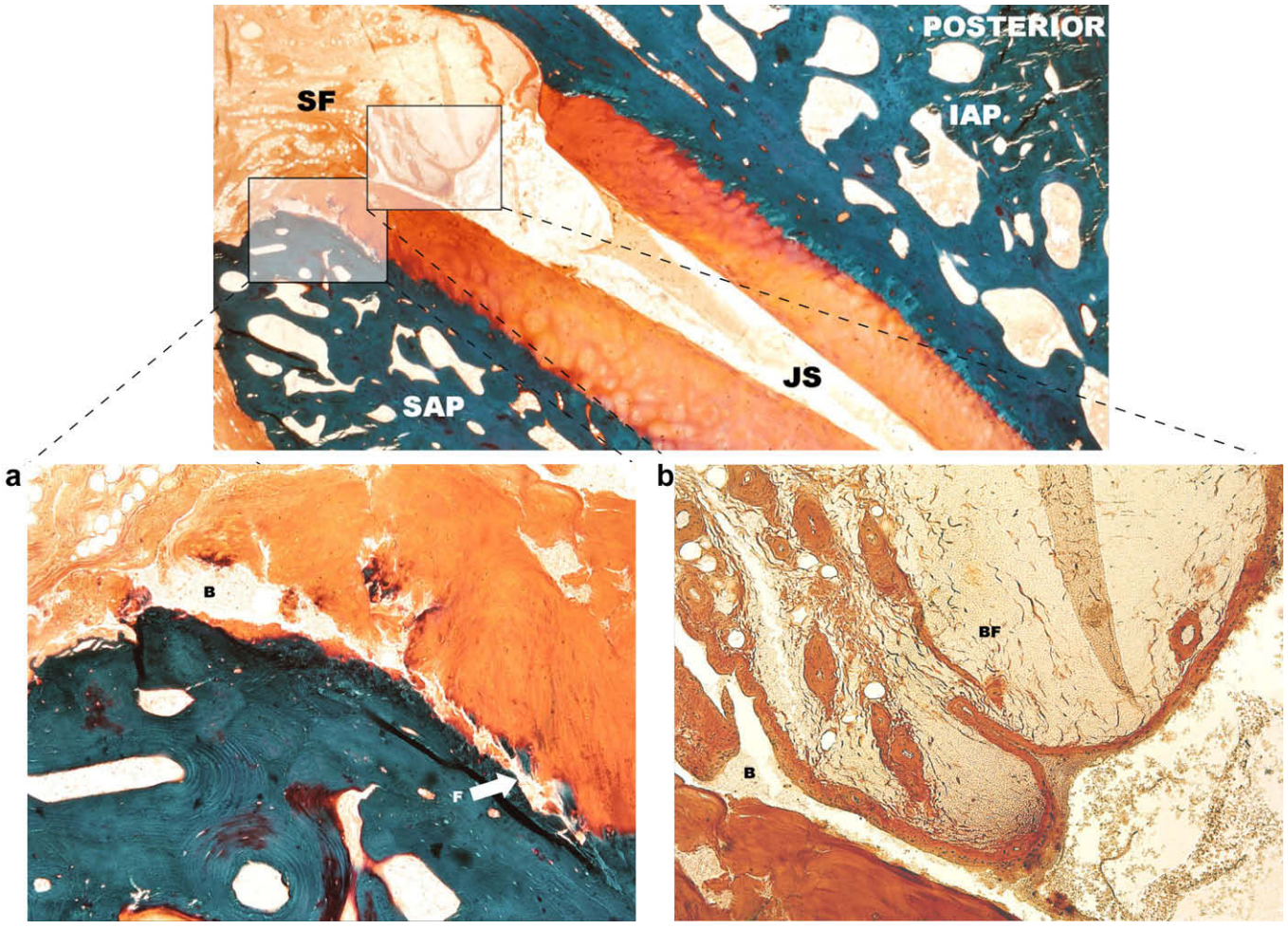

Fig. 3. Injuries to the cervical spine facet joint in a motor vehicle crash fatality: general overview of the joint, inferior articular process (IAP), superior articular process (SAP), synovial fold (SF), joint space (JS), original magnification ×1.25, Masson Goldner-Trichrome, and close-up of shaded areas illustrating bleeding in the fold (BF) and joint (B), and an osteochondral fracture (F), original magnification ×4, Masson Goldner-Trichrome.

of 3-mm thick slices do not allow reliable description of bleeding in the facet joints we find the previously reported results questionable.

This study identified significant injuries to both the anterior and posterior synovial fold. More posterior folds sustained injury and there was a highly increased risk of bleeding in these folds compared to the anterior counterparts after a fatal road traffic crash. Although no prior publications have reported specific subject based incidence rates for the occurrences or locations of bleeding in the folds, similar lesions have been reported previously.[7–11,19]

The presence of bleeding and disruption of synovial folds in a number of the control group subjects suggest that artefacts may be introduced at post-mortem. Despite good homogeneity (all cases were passenger car occupants, the age ranges were restricted to 20–49 years and the controls were non-traumatised) and detailed multifaceted evaluation of the study population, the gold standard chosen did not have the possibility of identifying all lesions. Facet fractures parallel to the line of sectioning would not necessarily appear on the histological sections and discrete fractures could potentially be present inside the 3-mm slices but outside the area of microtomal sectioning. Furthermore, prolonged ethanol fixation and staining of un-deplastified histological sections may have caused the poor visualisation of erythrocytes in lesions sites and thereby underestimation of bleeding.

Many people survive severe road traffic crashes and suffer consequently from a range of symptoms including neck pain. As the injuries identified in this study, which are in agreement with previously published findings, are non-fatal by definition, it is conceivable that similar pathology is present in some of these survivors. Furthermore, as all the injuries have nociceptive potential they can cause pain mediated through pain pathways. Similarly, the observed haemarthrosis may have clinical significance in the light of recent studies finding that even brief exposures to blood has detrimental long-lasting effect on the cartilage, by inhibiting the proteoglycan synthesis, thereby predisposing to premature degeneration.[22–25] Acute synovitis after haemarthrosis and/or cartilage damage after trauma has also been described in the literature,[22,25] which can also explain acute clinical manifestations of neck pain following relevant trauma. The possibility of degradation of cartilage matrix and subsequent development of degenerative changes in articular structures could correlate with diagnostic imaging findings of increasing osteoarthritis in road traffic trauma patients years after the injury,[26–28] although opinions are divergent.[27,29] The prevalence of injuries in cervical spine facet joints after survivable road traffic crashes have not been reported and the potential clinical implications are such injuries are inconclusive.

In this controlled study of people killed in passenger car crashes, non-fatal injuries to the lower cervical spine facet joints were a common finding, including facet fractures, haemarthrosis, disruption and bleeding in the synovial folds. The majority of lesions were not present on stereomicroscopic evaluation, and conventional autopsy procedures did not reveal any of the injuries. Hence, in selected cases where there is particular interest in microscopical details, specialized autopsy techniques should be encouraged in order to increase the sensitivity towards identifying discrete injuries, although they may have no influence on the cause of death.

The clinical implications of the lesions identified in this study are unknown, however, as imaging negative injuries may still be present there is an urgent need to improve the sensitivity of imaging modalities. At the present time we can offer no answer how to clinically identify such injuries and future studies are needed. Current knowledge suggests that discrete injuries similar to those identified in this study can be present in survivors from severe motor vehicle crashes.

Conflict of interest statement

None.

Funding

None declared.

Ethical approval

None declared.

Acknowledgements

The authors would like to acknowledge the original contributions to the conception of the design of the study from Professor DMSc. Flemming Melsen†, Institute of Pathology, Aarhus University hospital, the professional cooperation of the staff at the Institute of Forensic Medicine, University of Aarhus. The efforts of Rita Ullerup for the careful preparation of the histological sections are greatly appreciated. The study received unrestricted financial support from the European Chiropractors Union Research Fund Grant no. A.03-5, Switzerland, the Foundation for the Advancement of Chiropractic Research, Denmark and the University of Aarhus Research Foundation, Denmark. The support did not influence the study design, data collection, data interpretation, the writing nor the decision to submit the manuscript for publication.

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